Research Paper Medicine

A CASE REPORT OF MULTIPLE ORGAN DYSFUNCTION SYNDROME IN PARAQUAT POISONING

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ABSTRACT

A 17 year old female patient allegedly consumed a pesticide, which on enquiry turned out as 24% paraquat, around 20ml after a quarrel at home. Patient was given gastric lavage & supportive treatment. After 3-4 days patient had loose stools, difficulty in breathing, decreased urine output & blood stained vomiting leading to acute respiratory distress syndrome, hypotension, acute kidney injury and acute hepatitis. Patient was shifted to ICU. Then hemodialysis was done 3 times. There patient was on vasopressor & ventilator support for 3 days. That was followed by sudden cardiorespiratory collapse. When properly used, paraquat is widely used herbicide. This poisoning leads to death from multiple organ dysfunction and cardiovascular collapse within a week of intoxication.

KEYWORDS

Introduction

Paraquat is the trade name for N,N′-dimethyl-4,4′-bipyridinium dichloride, is the organic compound. It is classified as a viologen, a family of redox-active heterocycles of similar structure. This salt is one of the most widely used herbicides. It is quick-acting and non-selective, killing green plant tissue on contact. It is also toxic to human beings and animals.Paraquat poisoning leads to multiple organ dysfunction syndrome and death mostly due to respiratory failure. Treatment is mostly supportive care as no antidote available.

Case Report

A 17 year old female admitted to medical ward after consuming 20 ml of 24 % paraquat dichloride after a quarrel at home. Patient had episodes of vomiting immediately after ingestion and gastric lavage was done and managed supportively. Initial blood investigations were within normal limits. After 3-4 days patient had loose stools, decreased urine output and yellowish discolouration of urine and sclera. At that time S. Creatinine was found to be 10.8 mg%, B. Urea 130 mg%, S. Bilirubin 4.1 mg%. Blood gas analysis showed pH 7.4, pO2 90.6, pCO2 33, HCO3- 23.9, O2 sat 97%, PaO2/Fio2 . Electrocardiogram showed sinus rhythm. Ultrasonogram of the abdomen was normal. Hemodialysis was done thrice. S. Creatinine was in decreasing trend. After 2-3 days patient had blood in vomiting and difficulty in breathing and altered sensorium. Oxygen saturation dropped to 80%. Rapid sequence intubation was done and patient was put on ventilation. Patient's blood pressor was dropped and was put on vasopressor support. After 3 days of ventilator vasopressor support, patient succumbed.

Discussion

Paraquat is a quarternary nitrogen herbicide that is sprayed on unwanted weeds and other vegetations before planting crops. It is a fast-acting, non-selective compound, which destroys tissues of green plants on contact and by translocation within the plant.It is also toxic to human beings and animals. If ingested, paraquat induces a burning sensation of the mouth and throat, followed by gastrointestinal irritation, subsequently resulting in abdominal pain, loss of appetite, nausea, vomiting, and diarrhoea. Direct contact with paraquat solutions or aerosol mists may cause skin burns and dermatitis. Paraquat splashed in the eyes can irritate, burn, and cause corneal damage and scarring of the eyes. Irrespective of its route of administration in mammalian systems, paraquat is rapidly distributed in most tissues, with the highest concentration found in the lungs and kidneys. The primary injury caused by paraquat to mammalian systems occurs in the lung, where paraquat is accumulated through a process of active transport in the Clara cells and alveolar type I and II epithelial cells. The paraquat-induced lung injury is morphologically characterised by an early destructive phase, in which the alveolar type I and type II epithelial cells are damaged; and a second proliferative phase defined by alveolitis, pulmonary oedema, and infiltration of inflammatory cells. In addition to the lung, paraquat administration has been shown to injure other major organ systems, but to a lesser extent. Pathological changes have been observed in the liver, kidney, and heart at high doses; but death is usually associated with respiratory insufficiency injury. The mechanisms of paraquat toxicity involve the generation of reactive oxygen species and the oxidation of the cellular NADPH, the major source of reducing equivalents for the intracellular reduction of paraquat, which results in the disruption of important NADPH-requiring biochemical processes1. Treatment involves removal of ingested paraquat by immediately induced emesis or by gastric lavage in a health care facility. Clay (Fuller's earth) and activated charcoal are effective adsorbents.

Supplemental oxygen should be withheld unless the pO2 is less than 70 mmHg because oxygen may contribute to the pulmonary damage which is mediated through lipid peroxidation. Recent evidence regarding the use of immunosuppressive therapy with glucocorticoids and cyclophosphamide in the management of lung injury in patients with severe paraquat poisoning has been encouraging.

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